IS FASCICULAR TACHYCARDIA DIFFERENT FROM OTHER TYPES OF IDIOPATHIC VENTRICULAR TACHYCARDIA ?

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To test the hypothesis that patients with idiopathic (I) ventricular tachycardia (VT) showing a Right Bundle Branch Block (RB) configuration (C) and left axis (so called fascicular tachycardia) are a special type of VT 47 patients (pts) with IVT were analyzed. Data on exercise (Ex) testing (T) were available in 41, 24 hour ECG monitoring in 39, and programmed stimulation (PES) in 46 pts. In 25 pts isoproterenol (ISP) was used to facilitate the induction of VT. Eighteen pts had VT with a RBC, 9 with superior or left axis (LA) and 9 with intermediate or right axis (RA). Of 29 pts with a left bundle branch block (LB) C, 5 had LA and 24 RA.

	RBCLA	RBCRA	LBCLA	LBCRA	
Hx of VT during Ex	2/8 *	7/9	4/5	18/23	
VT induced by ExT	0/9 *	6/8	2/5	11/19	
> 30 VPB/hour	2/7 *	8/9	2/3	18/20	
VT Induced by PES	8/8 *	2/9	3/5	9/24	* p<0.05
VT Induced by ISP	0/2	2/7	2/3	6/13	
Termination by PES	7/7 *	1/2	2/3	4/7	
QRS width of VT	134±9*	166±24	156±6	157±27	
Recurrences	33%	22%	0	26%	

Pts with fascicular VT had less spontaneous VPB and VT was less often induced by ET and ISP than LBCRA pts, whereas VT was more often induced and terminated by PES. No differences were observed in the recurrence rate during 2.8±2.5 years of follow up. These data suggest that pts with fascicular tachycardia of the RBCLA type have a different electrophysiologic mechanism of VT.

MICROPOTENTIALS CONCEALED WITHIN THE SIGNAL-AVERAGED QRS COMPLEX - NEW INDEX OF HIGH-RISK IN MYOCARDIAL INFARCTION PATIENTS?

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Abnormal micropotentials generated in substrate of myocardial infarction (MI) might be, according to MI location, concealed within the entire QRS complex. The RMS amplitude (RMSA) was analysed cumulatively in 10 ms intervals in a "bidirectional" way - both "forward" from QRS onset and "reverse" from QRS offset (FC-RMSA and RC-RMSA, respectively). By this means, ventricular activation was studied in 32 healthy persons and 57 MI pts (>10 days following MI). The normal group is characterized by steep increases of both FC- and RC-RMSA. As abnormally depressed, flat RC-RMSA correspond to "late potentials" (LP), abnormally flat FC-RMSA increasing with delayed peaks (> 62 ms) were per analogiam designated as "early potentials" (EP). In MI pts, 2 typical patterns for each FC- and RC-RMSA can be recognized (presence+/absence- of EP and LP, respectively) and all 4 pattern combinations were found. The group characteristics with respect to micropotentials are as follows:

MI pts (total = 57)	EP+	EP-
anterior or combined MI	16	14
inferior or non-Q MI	3	24
LP present	8	13

These results support the hypothesis that all MI generate abnormal micropotentials, their timing within the QRS complex, however, corresponds to the time of activation of the infarcted area, i.e., early micropotentials predominate in anterior MI and late ones in inferior MI. The presence of both EP and LP identified in our study a very high-risk subgroup with 1) significantly ($p<0.02$) higher occurrence of ventricular tachycardia 2) low LVEF (<30%). Thus, detection of "concealed" early QRS micropotentials (in addition to "classic" LP) might further improve risk stratification in MI pts.

SIGNAL AVERAGED ECG IN PATIENTS WITH LBBB PATTERN VENTRICULAR TACHYCARDIA : CORRELATION WITH RADIONUCLIDE FOURIER PHASE ABNORMALITIES

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Signal averaged ECG (SAECG) and Fourier analysis (FA) of radionuclide ventriculography were compared in 41 pts with recurrent ventricular tachycardia (VT) with LBBB pattern and no evident heart disease. We have previously reported that FA accurately detects arrhythmogenic right ventricular dysplasia (ARVD) and quantifies RV wall motion abnormalities (RV-WMA) (RV to LV phase shift). SAECG was performed with bidirectional highpass filter (25 Hz). Group I: 20 pts without RV-WMA, group II : 21 pts with ARVD and significant RV to LV phase shift (109.7°±9.5° to 141.4°±42°, $p<0.01$). Results were:

	Group I	Group II	p
QRS (ms)	102±24.6	134.2±30	<0.001
LAS (ms)	27.1±11.7	47.9±27.8	<0.01
RMS 40 (V)	43.7±33.5	22.2±21.1	<0.02

Mean ± SD ; LAS : low amplitude signal duration; RMS 40: root mean square of last 40 ms.
Seven pts had mismatched results : normal FA with abnormal QRS (n=2) ; localized RV-WMA confirmed by cineangiography and normal SAECG (n=5). There was a positive correlation between RV to LV phase shift and QRS ($r=0.56$, $p<0.01$) and LAS ($r=0.44$, $p<0.05$). Conclusion : 1) SAECG and FA are in agreement in the detection of ARVD in pts with recurrent LBBB-VT ; 2) Abnormalities of SAECG correlate with the extent of RV-WMA. 3) Localized ARVD may have normal SAECG.

Polymorphous Ventricular Tachycardia in Patients with Normal Cardiac Function and QT Interval

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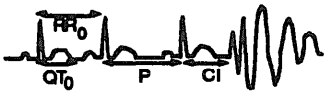
Thirteen pts presented with palpitations(4), pre-syncope(3), syncope(3), or sudden death(3) due to polymorphous ventricular tachycardia (PVT). Mean age was 38 yrs (20-64) and mean follow-up was 25 mo(1-56 mo). All had normal EKGs with mean QT=.40±.02 sec. There were no cardiac abnormalities found on echocardiogram (6), cardiac catheterization (7) or autopsy (1). Pts were on no medications during their clinical event, and electrolytes were normal. All episodes of PVT were analyzed in terms of preceding interval (P) and the relationship of the initiating coupling interval (CI) to the QT interval(QT_0).

RESULTS: 4 pts (Group I)had PVT reproducibly initiated by exercise. None were preceded by a pause, and PVT was initiated by late-coupled beats ($CI/QT_0>1.13$, mean 1.27±.12). Isoproterenol induced PVT in 3/4 and all 4 responded to chronic ß-blockade. Two pts (Group II) had PVT during episodes of coronary spasm and both responded to Ca blockers. PVT unrelated to exercise or coronary spasm occurred in 7 pts (Group III) and was pause dependent (P>RRo) in 5 (CI/QT_0 <1.12(0.97±.12). Aborted sudden death occurred in 3 Group III pts with the shortest CI/QT_0 (0.78-1.00). Group III pts responded to pacing, ß-blockade and Ca blockers alone, or in combination. One Group III pt discontinued ß-blockade and died suddenly. Programmed electrical stimulation was performed in 11 pts, and failed to provoke the clinical arrhythmia nor did monophasic action potentials (4 pts) show any early after depolarizations.



CONCLUSIONS: PVT can occur in pts with normal hearts and QT interval, and may be triggered by exercise, coronary spasm, or pauses. Patients with short coupling indices constitute a high risk subset. Correct diagnosis and appropriate therapy is associated with excellent prognosis.